AMERICAN INSURANCE ASSOCIATION

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June 13, 1980

TO THE TASK FORCE ON TORT LIABILITY
FOR CUMULATIVE TRAUMA AND LATENT INJURY

Re: (1) Revised Membership List
(2) Abel v. Eli Lilly

At the June 10th meeting of the above Task Force, Chairman Bailey suggested that a list of members which included addresses and telephone numbers be prepared and distributed. That list is attached. Please note that James J. Meyers of U. S. Insurance Group has been substituted in place of Robert J. Sullivan of Crum & Forster Insurance Companies.

We have received the final brief in the Abel v. Eli Lilly litigation. The brief is similar to the one which was distributed with the call for our June 10th meeting. If you wish to obtain a copy of the final brief, please let me know.

Very truly yours,

Dennis R Connolly

Dennis R. Connolly
Secretary

DRC/dt
Enc.

DEPOSITION
EXHIBIT
33
Lewis 5-19-92

34725

Diethylstilbestrol

The relationship between ingestion during pregnancy and adenocarcinoma of the vaginal and cervix in female offspring of mothers who took the drug was confirmed by epedimologic studies in the early 1970's. It was also discovered that non-cancerous lesions have developed in many of these female offspring. No evidence has yet been produced that these non-cancerous lesions progress to cancer. In fact recent reports indicate that the condition remisses with age. No confirmed evidence has yet been produced that mothers who took the drug and their male offspring are subject to an increased cancer risk.

DES was used by pregnant mothers in most cases to avoid miscarriages. The Department of Health and Human Services (former HEW) required labeling in 1971 contraindicating DES use for this purpose during pregnancy. Nevertheless, the drug is still prescribed for other conditions unrelated to pregnancy.

Studies indicate that DES usage during pregnancy varied regionally and annually. It was used more often in the East and Midwest, and less often in the South and West. Use during pregnancy probably reached a peak in the early 1950's and declined gradually thereafter.

Adenocarcinoma usually develops in daughters between the ages of 14 and 24. Peak incidence is at 19 years of age. The incidence rate has been estimated to be between .14 and 1.4 per 1000 exposed women.

Based on a number of studies it is estimated that there has been between 1.1 and 2.2 million daughters exposed in utero to DES. From this number between 1095 and 2226 adenocarcinoma cases are estimated to either have developed or are expected to develop between 1957 and 1993. From 1977 to 1993 between 143 and 311 cases are expected to develop.

Total payment for settlement and awards were made for two periods; adenocarcinoma cases developing between 1977 and 1993, and between 1970 and 1993. Average payment per case, after inflation adjustment, is taken to be $213,000.

Table III indicates the maximum potential payment for estimated future adenocarcinoma cases.

Table III
ESTIMATED MAXIMUM TOTAL PAYMENTS
FOR POTENTIAL ADENOCARCINOMA
CASES
1977 to 1993
(In 1980 Dollars)

Cases	Total Payments (In Millions)
143-311	$33-$71

To these cases currently contested DES cases should be added. But we cannot merely add, even if we knew the total number of cases, currently contested cases because a large number of women involved in these actions are suffering from adenosis, a health effect we are assuming will not engender any settlement or award. It is impossible at present to separate the adenosis cases from the adenocarcinoma cases. To provide an estimate of payments arising not only from future cases of adenocarcinoma but also from currently contested cases, the potential maximum payment for cases developing between 1970 to 1993 is shown in Table IV. Estimated loss adjustment expense is between $9.9 and $20.4 million.

Table IV
ESTIMATED TOTAL PAYMENTS
FOR ADENOCARCINOMA CASES
DEVELOPING FROM 1970 TO 1993
(In 1980 Dollars)

Cases	Total Payments (In Millions)
430-890	$91.5 - $189.5

1.0 Introduction

At the request of the Task Force on Tort Liability for Cummulative Trauma and Latent Injury the Research and Policy Development Department was requested to initiate a study of potential liability arising from a number of substances. Cummulative trauma and latent injury tort liability was identified as an important issue in liability insurance. Legal interpretation of products liability law is changing, as evidenced by the recent California Supreme Court verdict in Sindell vs. Abbott Laboratories and the Michigan appellate court decision in Abel vs Eli Lilly & Co. which propose an "industry-wide liability" concept in cases where "all defendants produce a drug from an identical formula and the manufacturer of DES which caused the plaintiff's injuries cannot be identified through no fault of the plaintiff." This new interpretation has enormous significance for the property-casualty industry not only in current retroactive cases where a harmful health effect has been determined to be caused by a condition which occurred in the past, but also for prospective cases where health effects have not yet become salient. There is apprehension that this legal interpretation will spread to other states and to liability claims arising from products produced in other industries. Because of the potential and substantial liability exposure which "industry-wide liability" opens, the Task Force decided that an attempt should be made to quantify some of these potential claims in order to assist the industry in evaluating future costs, to respond to outside questions and proposals, and to assist in determining industry positions on the issues.

Four current liability controversies were suggested for study: the asbestos, diethylstilbestrol (DES), Agent Orange and Love Canal issues. These four issues were divided into two groups; these where deleterious health effects have been confirmed (asbestos and DES), and these where health effects have not yet been confirmed in the medical and scientific communities (Agent Orange and chemicals disposed in the Love Canal). Because of the short period of time between initiation of the project and meeting of the Task Force, all four issues could not be handled in the detailed manner which is required to perform an adequate analysis. It was decided that only issues from group one would be analyzed, asbestos and DES.

2.0 ASBESTOS

2.1 Introduction

Asbestos is the generic name for a group of naturally occurring hydrated mineral silicates of the amphibole or serpentine series that are characterised by fibers or bundles of fine single crystal fibrils. Included in this definition are the following minerals: chrysotile, crocidolite, amosite and the fibrous varieties of anthophyllite, tremolite and actinolite. Chrysotile is of the serpentine variety, and the remaining minerals of the amphibole variety. More than 90 percent of all asbestos use. is of the chrysotile variety.

Because of its special physical properties asbestos has a wide range of product application. It is used in over 3000 different products for industry, commerce and the home. Its fire and heat resistant property makes asbestos particularly useful as an insulating material for air conditioning units, ducts, steam lines, pumps, and brake and clutch linings of automobiles. Asbestos has a high tensile strength so that it is also used as a reinforcing agent in cements and plastics for such products as cement pipes, and shingles. Some asbestos fibers, the anthophyllite and crocidolite fibers, are acid resistant so that they are used in products such as acid pumps, valves, and packings. Asbestos has also been used as a filtering material. During WWII it was used as a filtering material in gas mask cannisters. It was widely used to filter beer and other beverages.

Despite the useful product applications which have been made of asbestos since the modern beginning of the industry in 1880, evidence began accumulating that there were harmful health effects attached to its production and application. The first case of asbestosis was reported in England in 1906. The first reported case in the U.S. was made in 1930. A number of studies performed in the 1930's led investigators to relate asbestosis to asbestos exposure, if the exposure was long and occurred in an environment with high concentrations of dust. It was not until the 1960's that widespread recognition was made of the relationship between lung cancer and mesotheliomas, and asbestos exposure. More recently, asbestos exposure has been suspected to cause gastrointestinal and laryngeal cancer.

In 1938 the U.S. Public Health Service recommended limiting exposure to asbestos in industry to 5 million particles per cubic feet of air (176 particles/cc). This limit was formally recognized in 1964 as a guideline issued by the Bureau of Labor Standards. Not until 1972 did the newly established U.S. Occupational Safety and Health Administration adopt a legally enforceable standard of 5 fibers/cc longer than 5 microns. This was later lowered to 2 fibers/cc in 1976, and in 1975 OSHA proposed reducing it to .5 fibers/cc.

The following sections deal with the risk that exists for exposed workers, the estimated number of exposed workers and the estimated liability payments that may develop from such exposure.

2.2 Diseases

A number of health effects have been confirmed or suggested to develop from asbestos exposure. Considerable controversy has developed over the length of the fibers which seem to cause illness. Some researchers think that short fibers (those less than

5 microns in length) are the fibers which cause a fibrosis or induce a neoplasm. Since most exposure standards require that only the long fibers be counted, this controversy is of some interest. Some studies has concluded that it is the deposition of particles of approximately .5 to 5 microns in the alveoli and respiratory bronchioles that give rise to the group of diseases known as the pneumoconioses [1] The medical reason for concern for smaller particles is that larger particles will be filtered by various parts of the body. Nostril hair grating and the inpingement traps of the nasal passages will capture most of the larger particles (greater than 15 microns). The mucous coating of the trachea and branching bronchial passages will tend to trap more particles (above 10 microns). The inhaled air reaching the respiratory bronchioles and alveoli would contain the smaller particles (1 to 5 microns) [2]. This is an area where the medical, engineering and economic aspects of asbestos control confront each other. There is no consensus in the medical community that short fibers engender the various deleterious health effects. It is difficult to measure particles of such short length with readily available equipment. Complex and costly equipment is required. Therefore this is one of the reasons that exposure standards are usually defined in terms of long fiber counts.

2.2.1 Asbestosis

Asbestosis is a diffuse pulmonary fibrosis initiated by the inhalation of asbestos particles. It is a disease which is progressive and irreversible and leads to respiratory disability.

Though there appears to be no scientific evidence that asbestosis and lung cancer are interrelated, it has been shown in one study that approximately 50 percent of patients certified as suffering from asbestosis died of or with lung cancer [3]. There appears to be no regular correlation between the severity of asbestosis and the occurrence of mesothelioma. In fact, it is unusual to find significant asbestosis with pleural mesothelioma. However, asbestosis is frequently observed in association with peritoneal mesothelioma, which generally appears to be associated with heavier asbestos exposure [4].

The latency period for developing asbestosis seems to be somewhat shorter than that required to develop cancer. One study, which found asbestosis 11 times more common among pipe insulators in ship construction than in a control group, determined that asbestosis first appeared 13 years after initial exposure, and that the incidence was 38 percent after 20 years [4].

2.2.2 Respiratory Cancers

Lung cancer was first linked to asbestos exposure in 1935 when three cases were found at autopsy in asbestos textile workers. In 1949 in England autopsies of 225 persons, known to have asbestosis, found that 31 or 13.2 percent had lung cancer. This was not characteristic of other pneumoconioses. In 1954 a study found 10 times more lung cancer in persons employed 5 years or more in occupations involved in asbestos exposure than in a control group. Animal studies have confirmed these results [4].

Early diagnosis and surgical removal of lesions increases the survival rate. Irradiation, chemotherapy and immunotherapy may be helpful. It is generally regarded as a chronic incurable disease in which treatment may be

Among some groups of asbestos workers, approximately 20 percent of all deaths are caused by lung cancer where the expected proportion of deaths would only be about 5 percent [5].

There is also overwhelming evidence that asbestos exposure is related to pleural and peritoneal mesothelioma. Mesothelioma is a diffuse cancer that rapidly spreads over the surface of the lung, abdominal organs and heart. No effective treatment is presently available. Death occurs within 1 to 2 years of diagnosis. The first case report related to asbestos exposure appeared in 1933. However, it was not until 1960, with the publication of a series of cases in South Africa, that the association between mesothelioma and asbestos exposure was generally recognized. [5] Prior to the expanded use of asbestos, mesothelioma was a rare form of cancer. Estimates for certain occupational groups suggest that as many as 8 to 11 percent of deaths may be due to mesothelioma.

Cases of mesothelioma have also been reported in family members of workers exposed to asbestos, especially if contaminated clothing is laundered with other family clothes [2].

2.2.3 Digestive System Cancer

An excess risk of cancers of the digestive system attributable to occupational asbestos exposure has been suggested by a number of epidemiological studies. A major problem with these studies has been the inclusion of peritoneal mesothelioma cases among all observed cases, making it difficult to document an increased risk of any one digestive system cancer independent of mesothelioma.

In an occupational cohort mortality study where peritoneal mesothelioma cases were separated from other digestive system cancers, excess cancers of some sites were observed. Among 433 amosite asbestos factory workers who were first employed between 1941 and 1945, there were 11 deaths from stomach cancer by 1971 when 4.58 were expected, 15 deaths from cancer of the colon or rectum when 7.05 were expected, and none from cancer of the esophagus, when 1.23 were expected [4].

2.2.4 Laryngeal Cancer

The evidence linking asbestos and laryngeal cancer is highly suggestive. In a study of 119 patients with squamous carcinoma of the larynx, 33 with laryngeal cancer but only 3 age-sex matched controls gave a history of occupational exposure to asbestos [4].

2.3 Latency Period and Dose-Response Relationship

The latency period of asbestos related cancers tend to be long and the range within which the cancer develops tends to be wide. From all available evidence, the period between first exposure to asbestos and death from lung cancer appears to be related to intensity of exposure. Among workers in an English asbestos textile and insulation material factory, an excess mortality from lung cancer was demonstrated following a latency period of 15 years for those with heavier exposures, whereas an excess did not appear until 25 years from onset of exposure for those whose exposure was less intense. Fifteen years is probably the minimum latency period for asbestos related lung cancer. In one study the peak increase occurred about 30-35 years after onset of exposure [4]. In a study reported in reference [4] death from mesothelioma occurred more than 25 years after first exposure in 85 percent of the mesothelioma cases, with a range of 3.5 to 53 years. Another report showed a mean latency period of 37 years.

There is some evidence suggesting that dose, as measured by severity of exposure and duration of employment, relates to rates of cancer. As reported in reference [4] one study concluded that within an age cohor for which data were most accurate there was an increased mortality due to lung cancer with increasing duration of employment. Another study, whose results are shown in Table 2.1, showed an increase risk of respiratory cancer with increasing exposure. Exposure is defined as the product of dust levels in millions of particles per cubic foot of air and years of exposure

TABLE 2.1
Deaths From Respiratory Cancer By
Cumulative Dust Exposure

Total Dust Exposure (mppcf - yrs)	Respiratory Cancer Deaths		
	Observed	Expected	SMR*
Under 125	15	9.0	166
125-249	12	4.8	250
250-499	17	5.2	326
500-749	9	1.8	500
750 and Over	5	.9	535

Source: Reference (6)

* SMR is the standard mortality ratio defined as observed divided by expected deaths. It measures the increase risk.

The excess risk of respiratory cancer was close to 4 times greater in the highest exposed group compared to the lowest exposed group. While most researchers are in agreement that risk does increase with magnitude of exposure, either amount and/or duration, they also agree that there does not seem to be a lower threshold value (LTV), i.e. a value below which cancer does not develop. Therefore there does not seem to be an LTV in terms of duration of employment or level of dust exposure for occupational exposures.

2.4 Incidence

Incidence rates of cancer vary among published studies. As reported in reference [7] a seven-fold excess of lung cancer was found in a group of insulation workers. Another study reported a 4.4 times increase in risk of respiratory cancer among retired production and maintenance employees in the asbestos industry. In the asbestos cement industry a 6.1 times greater risk was found. Among workers in the mining and milling industries between 1947 and 1961 cancer mortality among male workers was 1.6 times greater than in the general male population. The lung cancer rate for males was twice that of the general male population. Another study, of amosite insulation production workers, reported a three-fold increase in lung cancer among workers with less than 3 months of employment and among workers with less than 1 month of employment a 2.25 increase. A study performed by the National Cancer Institute (NCI), the National Institute of Occupational Safety and Health (NIOSH) and the National Institute of Environmental Health Science (NIEHS) used a range of 1.5 to 12 with an average of 6.6 for the excess risk of lung cancer and mesotheliomas [8].

2.5 Synergism

There is strong evidence that smoking and asbestos are synergistically related. There is also evidence that the relationship is not additive but multiplicative. An additive relationship would hold that with an excess risk from asbestos exposure alone of 6 and an excess risk from smoking without asbestos exposure of 6, asbestos exposure and smoking would produce an excess cancer risk of 12. A multiplicative relationship will produce an excess risk of 36.

In a study of the combined effects of asbestos exposure and smoking the smoking habits of 1,334 male and 482 female asbestos factory workers were examined in relation to mortality from lung cancer. Among smokers with severe asbestos exposure 41 lung cancer deaths were observed, when only 11.3 were expected for smokers from the general population. Among 161 never-smoking asbestos workers with severe asbestos exposure, 1.7 lung cancer deaths were observed, when only .2 were expected for non-smokers in the general population [9].

In the general population, cigarette smokers have a 10-15 fold excess risk of lung cancer. One study observed an 8-fold excess of lung cancer among smoking asbestos workers compared with smokers in the general population, but the excess was 92-fold compared to the general population of non-smokers [10]. Though the multiplicative hypothesis is held by some researchers, it has not been definitively established.

There is very little evidence that cigarette smoking increases the risk of developing pleural mesothelioma after asbestos exposure, and no evidence exists that smoking increases the risk of peritoneal mesothelioma.

2.6 Methodology For Estimating Liability Payments

A methodology was developed to estimate the number of cancer deaths which have occurred since 1951 and projected to occur to 1995 from occupational exposure to asbestos, and to estimate the potential liability payments which cases developing between 1977 and 1995 may engender. Since the latency period for developing an asbestos related disease is long, the year 1951 was selected to capture most of the potential cases, and to avoid double-counting most of the WWII shipyard workers. Older employees in 1951 and earlier years were not likely to be alive at the start of our estimating period in 1977. For these reasons it was not thought worthwile to start at an earlier period. This methodology attempts to take into consideration three major limitations inherent in previous methodologies. Most previous exposure figures were derived by subtracting all clerical, supervisory, administrative and maintenance employees in a plant or industry in which asbestos is found from the total number of employees. The remainder, primarily production workers, was assumed to be the population at risk. This procedure usually overestimates the number of workers exposed. It does not consider the possibility that groups of production workers are exposed in different degrees to asbestos. The second limitation of previous methodologies is that they consider only currently employed workers. They do not consider workers who were employed for a time but, for one reason or another, left the industry. The third limitation of these methodologies is that they do not consider the duration of exposure and magnitude of exposure of former or current workers. As will be seen, an attempt is made to overcome these limitations, and include all three variables in the analysis. Our analysis will account not only for current workers, but also former workers who are no longer exposed. For each of these groups a measure of duration of exposure and magnitude of exposure will be developed.

Our methodology can be divided into three major steps. These steps are to estimate

- the number of exposures
- the number of cancer and asbestosis cases which develop from exposures and their distribution over the years
- the potential liability payments

2.6.1 Exposures

Within the time constraints of the study it was not possible to analyse asbestos exposure in every industry in the United States. It became necessary to identify those industries which have the largest number of people occupationally exposed to asbestos. In 1972 the National Institute of Occupational Safety and Health (NIOSH) initiated a two year field study to determine the extent of worker exposure to chemical and physical agents [11]. A team of 20 engineers surveyed a sample of 5000 plants in 66 different two digit Standard Industrial Classifications (SIC), located in 67 Standard Metropolitan Statistical Areas (SMSA). Approximately 200 hazards were identified, one of which was asbestos. Of the information recorded during the survey, the surveyor noted the number of workers exposed to the substance, and the daily duration of exposure, classified as full-time or part-time exposure. Full-time exposure included all workers exposed to a substance more than 4 hours a day.

The 10 two digit SIC manufacturing industries with the largest number of employees exposed to asbestos both full-time and part-time were identified and used in further analysis. This screening produced 13 manufacturing industries because some industries were in the top 10 in both categories. Contract construction was also included in the group because of the large number of exposed workers in the industry, making a total of 14

industries. These 14 industries and the estimated number of exposed workers are shown in Table 2.2. They had 90.7 percent of the full-time exposed workers estimated in the NIOSH study, 63.5 percent of the part-time exposed workers, and 65 percent of all exposed workers. The last two columns of Table 2.2, which were computed from data in the study, show the percentage of workers exposed full-time and part-time to asbestos relative to all workers in the industry. These percentages are used later in the analysis.

The NIOSH study is the most comprehensive analysis of hazardous exposure by industry classification, but unfortunately it applies only at a point in time, 1972 to 1974. Since we are interested in estimating exposures over time, from 1951 to the present, it was necessary to apply the percentages of Table 2.2 to employment figures in each year from 1951 to 1979. This necessitates assuming that the percentage of exposed workers estimated in 1972-1974 also hold for other years. The major factors which would affect the percentage of workers exposed are likely to be the types of machines used, the space relationship of workers to machines, and substitution of other materials for asbestos in the production process. These factors change only slowly over time in an industry. For example, the space relationship between men and machines depends on the physical plant size and layout. Replacement and expansion of old plants occurs only slowly in an industry. Therefore our use of the 1972-1974 percentages are likely to be severely biased only in the early years of our analysis. This result is somewhat mitigated because our final figures of asbestos induced cancer cases are estimated only from 1977 to 1995.

Table 2.2
Number and Percent of
Exposed Workers by Industry
and Type of Exposure
1972-1974

Industry	Total Exposed	Full-time Exposed	Part-time Exposed	Employment	Percent Full	Percent Part
Contract Construction	430,570	2,512	428,058	2,532,382	.1	16.9
Textile Mill	8,256	3,432	4,824	232,122	1.5	2.1
Apparel	87,929	35,573	52,356	918,752	3.9	5.7
Lumber & Wood	6,009	1,466	4,543	161,332	.9	2.8
Furniture & Fixture	15,043	818	14,225	295,253	.3	4.8
Printing & Publishing	55,741	437	55,304	1,239,262	.04	4.5
Chemical & Allied	23,314	429	22,885	966,199	.04	2.4
Stone, Clay & Glass	60,299	10,274	50,025	700,104	1.5	7.1
Primary Metal	61,878	2,107	59,192	1,347,780	.2	.4
Fabricated Metal	36,643	3,386	33,263	1,350,047	.3	2.5
Machinery except Electrical	89,423	570	88,853	1,539,734	.04	5.8
Electrical equipment	38,552	4,498	34,054	1,501,365	.3	2.3
Transportal equipment	86,195	1,531	84,664	1,224,441	.1	6.9
Miscellaneous Manufacturing	18,032	8,092	9,939	384,114	2.1	2.6

Source: Reference 11

Using these percentages we can estimate the number of exposed workers in each industry classified into two categories, full-time and part-time exposure.

Our next step was to provide an estimate of the number of workers who left each industry in each year from 1951 to 1979. The result of this step will provide not only an estimate of the number of workers who were removed from asbestos exposure, but also, by performing the computations each year, the number of years of exposure of each person. To accomplish this task we made use of annual accession and separation rates, and annual employment figures by industry, published by the Bureau of Labor Statistics. The accession rate is defined as the number of persons hired or rehired per 100 persons employed in the industry, and the separation rate is defined as the number of persons who quit, were laid-off or fired, or were separated through retirement, death or disability per 100 persons employed in the industry. To understand the procedure, see Appendix A for a full explanation.

The tables also provide an estimate of 1979 employment by number of years employed. We, therefore, can develop an estimate of asbestos exposure for currently employed workers by applying the percentages of Table 2.2 to 1979 employment. To the totals of currently exposed workers and formerly exposed workers, we will add an estimate of WWII shipyard workers.

2.6.2 Incidence

Once summary tables for former workers were developed, workers exposed full-time and part-time were separated into a high risk group and a low risk group. Incidence rates were then applied to each risk group and the estimated cancer cases were distributed over time based on the range of latency periods provided in the literature.

All workers exposed part-time were placed in the low risk group and all workers exposed full-time 5 years or less were placed in the low risk group. All other workers exposed full-time were placed in the high risk group. A sensitivity analysis based on an equivalency measure between workers exposed full-time and workers exposed part-time can be computed. Such a measure will be based on hours of exposure and will be presented at the Task Force meeting.

To provide a lower bound on the analysis, a second estimate was computed based on assuming all workers, regardless of exposure, are in the low risk group.

Reference [8] states that the cancer incidence of less exposed workers is 25 percent of that of highly exposed workers. This study uses a risk ratio range of 1.5 to 12 and an average of 6.6 for lung cancer and mesothelioma. We have used a risk ratio of 6.6 for our high risk group and 1.5 for our low risk group, approximately 25 percent of the incidence of the high risk group.

To determine excess cancer cases the formula (R-1) IN is used in reference [8] where

R is the risk ratio
I is the general population cancer rate per 100,000 adjusted for people 20 years and older
N is the number in the exposed population

By using this formula the number of excess cancers per year attributable to asbestos exposure over and above what would be expected can be computed. We have slightly changed this formula in our computation, and used RIN. This gives the total cancer cases attributable to asbestos exposure. In a normal medical or courtroom situation it would be impossible to determine that an asbestos worker who has cancer developed it not from his asbestos exposure but because his lifestyle predisposed him to develop it or that he was part of some statistical population which would be expected to develop it. Therefore all the information necessary to estimate asbestos induced cancer has been established. R is either 6.6 or 1.5. I is 116 for lung and mesothelioma and N is the number exposed. For former workers each element in our summary table is multiplied by 116 and 6.6 if the person is in the high risk group, or 1.5 if the person is in the low risk group.

These cases must then be distributed over the range of latency periods for the two different risk groups. A latency period of 15 to 40 years from first exposure was used for the high risk group and 25 to 40 years for the low risk group. For lack of a better distribution function, cases were uniformly distributed over the years.

2.6.3 Liability Payments

Total payments and liability payments were taken from the ISO Products Liability Closed Claim Survey, published in 1977. Average payments for bodily injury claims for various types of injuries are reported in this study, as of March 15, 1977. Payments prior to 1977 were trended for severity to 1977.

Average total payments for injury due to cancer averaged $166,883, and for injury associated with asbestos, $169,956. Since there is not a large difference in there figures, $168,000 was used for lung cancer cases. Payments for death claims average somewhat less than for cancer cases, $132,871. Most mesothelioma cases are likely to be made for death claims. Our final incidence figures do not separate lung cancer cases from mesothelioma cases, but we know that death from lung cancer is approximately 3 times death from mesothelioma. Based on this relationship, a weighted average of $159,000 was used as our estimate. Average Payments of $168,000 were used for asbestosis cases.

Each of these averages were inflation adjusted to 1980. Between March, 1977 and May 1980, the Consumer Price Index rose 37.5 percent and the Medical Cost portion of the Consumer Price Index rose 34 percent. A figure of 35 percent was used for our inflation adjustment.

2.7 Estimated Liability Payments

This section will bring together the three populations at risk from asbestos exposure and estimate the potential liability payments arising from the three types of illnesses caused by such exposure. The three populations are:

- currently non-exposed workers who were exposed to asbestos in the past in 14 industries
- currently exposed workers in these 14 industries and
- WWII shipyard workers

The three illnesses are:

- lung cancer
- mesothelioma and
- asbestosis

Gastrointestinal and laryngeal cancer has not been included in the estimates because the evidence does not appear to be as well established as it does for the other illnesses. This can be easily included if the task force finds it appropriate.

It should also be noted that there is probably some slight double accounting between two of our classes. Shipyard workers are a sub-classification of the transportation equipment industry, one of our 14 industries. In 1950, the start of our employment analysis, some shipyard workers were probably holdovers from the WWII period. It is not possible at this time to eliminate those numbers from our transportation equipment sector. Relative to the total number of workers in our analysis

and number of cancer cases estimated for shipyard workers, this double accounting should be small.

Tables 2.3 and 2.4 show the estimated number of lung cancer and mesothelioma cases by year of diagnosis, based on different risk ratios. Cases are estimated from 1977 to 1995 for both currently employed workers and past workers in the 14 industries. Estimates begin in 1977 because of the 3 year statute of limitation. The statute of limitation is assumed to begin running at diagnosis of the illness. At least three court cases have established that the statute of limitation begins to run when harm or impairment has manifested itself, or at the time the injured party should have discovered the illness.

In Karjala vs. Johns - Manville Products Corp., 523 F. 2d 155, Minn., 8th Cir. (1975), the court stated that a claim must be asserted when the impairment or harm has manifested itself in a way that supplies some evidence of causal relationship to the manufactured product. The time that impairment manifests itself is a jury question. In Harig vs. Johns - Manville Product Corp., 284 Md. 70; A. 2d 299 (1978), the court held that in latent diseases the cause of action accrues when plaintiff discovers or by a reasonably diligent search could have discovered that there was a cause of action. In Nolan vs. Johns - Manville Asbestos and Magnesia Materials Company, Appeal from the Circuit Court of Cook County, No. 77-724 (24 July 1979), the court suggested that where slow-developing industrial diseases are involved, the statute of limitations runs from the time that the diseased party discovered or should have discovered (1) his illness and (2) that the illness was the result of the wrongful conduct of another. The court concluded that this issue requires more than a mere computation of calendar days and that it is an issue of fact for the jury to determine [2]

Table 2.3
Estimated Lung Cancer and
Mesothelioma Cases
Based on High Risk and Low Risk Groups

1977-1995

Year Diagnosed	From Current 1979 Employment	From Previous Employment	Total
1977		1670	1670
1978		1748	1748
1979		1761	1761
1980	1044	1865	2909
1981	1080	1966	3046
1982	1118	2011	3129
1983	1202	2021	3223
1984	1250	2141	3391
1985	1289	2011	3300
1986	1369	2015	3384
1987	1458	2058	3516
1988	1528	2171	3699
1989	1033	2203	3236
1990	1109	1774	2883
1991	1072	1793	2865
1992	1088	1683	2771
1993	1113	1645	2758
1994	1125	1616	2741
1995	1123	1613	2736
TOTAL	19,001	35,765	54,766

Table 2.4
Estimated Lung Cancer and Mesothelioma Cases Based on all Workers in Low Risk Group

1977 - 1995

Year Diagnosed	From Current 1979 Employment	From Previous Employment	Total
1977		1189	1189
1978		1255	1255
1979		1251	1251
1980	716	1347	2063
1981	741	1415	2156
1982	764	1451	2215
1983	827	1465	2292
1984	868	1587	2544
1985	895	1456	2351
1986	950	1474	2424
1987	991	1522	2513
1988	1041	1651	2692
1989	648	1689	2337
1990	734	1410	2144
1991	714	1435	2149
1992	785	1345	2130
1993	780	1324	2104
1994	803	1322	1225
1995	808	1335	2143
Total	13,065	26,923	39,988

While individual juries may find the discovery of an illness some time other than at diagnosis, it is likely that in most cases diagnosis will indicate discovery.

To Tables 2.3 and 2.4 we can add the estimated number of asbestosis cases in the 14 industries. There were no studies found during the course of our analysis which determined an overall incidence rate for asbestosis. Only rates for a few specific occupations have been determined. One cannot just add an estimate for asbestosis cases to the lung cancer and mesothelioma cases. This procedure would result in double accounting. As we saw in section 2.2.1, 50 percent of people certified as showing asbestosis died of or with lung cancer. Therefore asbestosis cases should equal twice the number of lung cancer cases. Table 2.3 shows 54,766 lung and mesothelioma cases estimated to occur between 1977 and 1995. But there is no evidence that mesothelioma and asbestosis are related, therefore mesothelioma cases must be removed from the 54,766 cases. As we have also seen in section 2.2.2, lung cancer deaths occur in approximately 20–25 percent of all deaths of asbestos workers, and mesothelioma deaths in 7–10 percent of all deaths of asbesots workers, a ratio of approximately 3 lung cancer deaths to 1 mesothelioma death. Using this relationship we can approximately separate the lung cancer cases from the mesothelioma cases and determine the total asbestosis cases, independent of cases where lung cancer and asbestosis appear together.

Number of asbestosis cases: = .75 times total lung and mesothelioma cases times 2

Using this formula we derive approximately 82,000 asbestosis cases between 1977 and 1995. But 50 percent of these asbestosis cases would also have lung cancer, therefore the estimated number of cases

where asbestosis appears alone is approximately 41,000. The other asbestosis cases are counted with the more serious lung cancer cases. Using the same procedure in Table 2.4 produces 30,000 asbestosis cases.

The final cases to add to our present total are WWII shipyard workers. Only a very rough estimate can be computed for this group because statistics of its age distribution and employment history is lacking in published reports. In fact the only figure mentioned in a number of reports is that 4.5 million persons worked in shipyards during WWII [8]. But based on some crude, but we feel reasonable assumptions, we can derive an estimated number of cancer cases for these workers. We can start with two facts. Women were a large percentage of the work force during WWII and the average age of male workers was probably higher than usual because young men were in the armed services. Reference [8] states that of the 11 million workers believed to have been exposed to asbestos since the beginning of WWII, 4 million workers were heavily exposed, or about 36 percent. Applying this number to the 4.5 million shipyard workers we derive 1.6 million heavily exposed shipyard workers. If we assume that 50 percent were males and 50 percent female, we have 800 thousand in each group. It is also likely that the male workers were older than female workers on average. If we assume that male workers were between 35 and 60 years of age and female workers between 25 and 50, and that the age distribution was uniform, we can estimate that there were 32 thousand male workers at each age between 25 and 50. With a minimum latency period of 15 years for high risk exposure, only some male workers would have lived to the year 1955, assuming all workers started in the shipyards in 1940. We can make the reasonable assumption that only males 50 or younger in 1940 lived to the start of latency in 1955. This amounts to about 480 thousand male workers. Using a lung cancer and mesothelioma rate of 32 percent of

all deaths, there would be about 154 thousand cases or 6,100 per year. The latency would end in 40 years in 1980. Therefore male cases would add 5,100 in each year from 1977 to 1980. All female workers are assumed to reach the start of latency in 1955 since the maximum age assumed is 50. Applying the 32 percent lung cancer and mesothelioma death rate to the 800 thousand female workers produces 256 thousand cases or about 10,200 per year. Table 2.5 summarizes these results. Only lung cancer and mesothelioma cases are computed, since asbestosis cases should have made their appearance long before 1977.

Table 2.5
Estimated Lung Cancer and
Mesothelioma Cases in WWII
Shipyard Workers
Based on High Risk Rate

1977 to 1980
(In 000)

Year	Male	Female	Total
1977	6.1	10.2	16.3
1978	6.1	10.2	16.3
1979	6.1	10.2	16.3
1980	6.1	10.2	16.3
Total	24.4	40.8	65.2

To develop an estimate of lung cancer and mesothelioma cases in WWII shipyard workers based on a low risk incidence rate, a minimum latency of 25 years and an incidence rate of 8 percent was used. Eight percent is 25 percent of the 32 percent high risk rate, a relationship used in reference [8]. Only 320 thousand male and 640 thousand female workers were assumed to live to the start of latency in 1965, i.e., all workers who were 45 or younger in 1940. Table 2.6 shows the result of this computation.

Table 2.6
Estimated Lung Cancer and
Mesothelioma Cases in WWII
Shipyard Workers
Based on Low Risk Rate

1977 to 1980
(In 000)

Year	Male	Female	Total
1977	1.7	3.4	5.1
1978	1.7	3.4	5.1
1979	1.7	3.4	5.1
1980	1.7	3.4	5.1
Total	6.8	13.6	20.4

We can now add all of our classifications together in a summary table. This is done in Table 2.7.

Table 2.7
Estimated Potential Number of
Claims Due to Asbestos Exposure

1977 to 1995
(In 000)

Illness	Population	Potential Number of Claims Based On Low Risk Only	Low Risk High Risk
Lung Cancer & Mesothelioma	Current and past employees[1]	40.0	54.8
Asbestosis	Current and past employees[1]	30.0	11.0
Lung cancer & mesothelioma	WWII shipyard workers	20.4	65.2
Total		90.4	161.0

[1]Calculated from the 14 industries with largest number of employees exposed to asbestos. They included 65 percent of employees exposed in all industries.

Table 2.8 shows the estimated range of total payments, arising from either a settlement or an award, due to asbestos exposure in 1980 dollars. An inflation factor of 35 percent was used to adjust average payment for lung cancer and mesothelioma cases from $159,000 to $215,000, and asbestosis cases from $168,000 to $227,000. Average payment includes payment not only from product liability sources but from other sources as well.

Table 2.8
Estimated Maximum Total Payments
Due to Asbestos Exposure

1977 to 1995
(In 1980 Dollars)

Illness	Number[1] (In Thousands)	Average Payment (In Thousands)	Total Dollars (In Billions)
Lung cancer & mesothelioma	60.4 - 120.0	$215.0	$13.0 - $25.8
Asbestosis	30.0 - 41.0	227.0	6.8 - 9.3
Total			$19.8 - $35.1

[1]Calculated from 14 industries with largest exposure and WWII shipyard workers.

The ratio of product liability payments to total payments for asbestos settlements and awards is not provided in the ISO study. But for payments made for all of the various injury claims in the $100,000 to $500,000 range product liability payments averaged 84.1 percent of total payments in the ISO closed claim survey. Therefore the estimated product liability payments arising from asbestos induced diseases is between $16.7 and 29.5 billion.

Table 2.9
Estimated Maximum Product Liability
Payments for Asbestos Induced Diseases

Number of Cases (In Thousands)	1977 to 1995 (In 1980 Dollars) Total Payments (In Billions)	Ratio Product Liability Payments to Total Payments	Product Liability Payments (In Billions)
90.4 - 161.0	$19.8 - $35.1	.841	$16.7 - 29.5

These figures should be considered maximum limits for the 14 industries and WWII workers analyzed for two reasons. Not all of the expected cancer and mesothelioma cases will be handled through the product liability system. Some cases may be handled by workers' compensation, while other cases may not even be reported because of death of the workers and ignorance of possible legal remedies by the decedents' families. To adjust for the workers' compensation system, we would ideally like to have an estimate of the percentage breakdown of cases handled through the product liability system compared to the workers' compensation system. Unfortunately, such a figure was not readily available for this analysis. But if we can assume that the probability of a case being handled through workers' compesnation is as likely as it is to be handled through product liability, we can derive an estimate of potential product liability payments. This is shown in Table 2.10.

Table 2.10
Estimated Likely Product Liability
Payments Due to Asbestos Exposure
1977 to 1995
(In 1980 Dollars)

Illness	Total Cases[1] (In Thousands)	Probability Handled Through Product Liability	Average Product Liability Payment (In Thousands)	Total (In Billi
Lung cancer & mesothelioma	60.4 - 120.0	.5	$180.8	$5.5 - $10
Asbestosis	30.0 - 41.0	.5	190.9	2.9 -
TOTAL				$8.4 - $1

[1]Based on 14 industries having 65 percent of all exposed members and WWII shipyard workers.

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These figures do not include allocated loss adjustment expense. For payments over $100,000 in all types of liability claims, the allocated loss adjustment expense averaged 10.8 percent of payments in the closed claim survey. Therefore, loss adjustment expense can be estimated to be between $900 million and $1.6 billion.

Table 2.11
Estimated Likely Product Liability
Payments and Loss Adjustment
Expense Due to Asbestos Exposure

1977 to 1995
(In 1980 Dollars)

	(In Billions)
Product liability payments	$8.4 - 14.7
Loss Adjustment expense	.9 - 1.6
Total	$9.3 - $16.3

Finally, it should be noted that the figures have not been adjusted for possible synersistic effects, in particular smoking. The establishment of the synersistic relationship between smoking and asbestos exposure may reduce the dollar costs of settlements and awards, if the courts find contributory negligence on the part of smoking workers.

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3.0 DES

3.1 Introduction

Diethylstilbestrol (DES) is a synthetic estrogen which was first produced in 1938 by Professor E.C. Dodds and his associates. From about 1941 to 1971 it was prescribed as a remedy to women with a history of threatened pregnancies, though in some cases it was prescribed for pregnant women without such a history. In the early 1950's researchers at the University of Chicago reported that the drug was ineffective in preventing miscarriages, and its use for this purpose gradually declined. In 1970 doctors in Chicago and New England reported a sudden increase in a rare form of vaginal and cervical cancer, clear-cell adenocarcinoma, in adolescent females. Prior to this time, adenocarcinoma of this type was practically non-existent in young women. Epidemiologic studies confirmed an association between DES and ingestion of the drug during pregnancy by mothers of these adolescents. In November, 1971 the Food and Drug Administration required manufacturers to label the drug as contraindicated for use in the prevention of miscarriages. DES, nevertheless, continues to be used in a number of ways; as an estrogen replacement in cases of hormone deficiency, prevention and treatment of certain menopausal-related problems, treatment in selected cases of advanced breast and prostate cancers, suppression of lactation in new mothers who do not breast feed their infants, and as a feed additive for cattle and sheep. Of the synthetic estrogens, DES was the most widely prescribed and was used in the largest amount, but there are many similar drugs that may have been prescribed to pregnant women (see Appendix B). HHS* has noted in their Task Force report [1] that there is no evidence that natural or other synthetic

* HEW is now called the Department of Health and Human Services.

estrogens differ substantially in their harmful or toxic effects.

The following sections deal with the risk that exists for exposed daughters, mothers, and sons, the estimated number of exposed daughters and the estimated liability payments that many develop from such exposure.

3.2 Diseases

Medical tests have been performed not only on the daughters exposed in utero to the drug but also on sons exposed in utero, and on the mothers themselves. A number of health effects have been either confirmed or suggested to exist in these groups.

3.2.1 Daughters

The confirmed or alleged health effects on daughters have been the most widely publicized. These effects include adenocarcinoma of the vagina and cervix, vaginal epithelial changes (VEC), infertility, and adverse pregnancy outcomes. As has been mentioned, adenocarcinoma has been epidemiologically established in a number of medical studies [2], [3], [4], and will be discussed in Section 3.3

In order to study the incidence and history of genital changes in DES daughters the National Cancer Institute funded in 1974 a cooperative project of four major medical research centers; Baylor College of Medicine, Massachusetts General Hospital, the University of Southern California and the Mayo Clinic. This project has come to be known as the DESAD (DES - Adenosis) Project. DESAD has recently completed major studies dealing with the incidence of VEC (vaginal epithelial changes). In these studies VEC is defined as any mucosal change of the vagina observed macroscopically on the basis of colposcopic examination or iodine staining. Vaginal adenosis, a non-malignant glandular tissue, is a sub-category of VEC. The DESAD project consists of 3339 participants who were known to be or suspected of having been exposed

in utero to DES. Some were identified through review of prenatal records, other participants requested entry into the program, and still others were referred by a physican.

Two studies dealing with VEC in exposed daughters have been completed by DESAD [5] [6]. Of the 1275 participants identified by prenatal records 34 percent exhibited VEC, and 37 percent of the participants with VEC who had biopsies performed had adenosis. Regional differences in the extent of VEC in the participants was also found. The frequencies at the four medical centers were: 63 percent at Massachusetts General Hospital, 50 percent at the University of Southern California, 26 percent at the Mayo Clinic and 24 percent at the Baylor College of Medicine. Variations in total dosage ingested by mothers, duration of exposure, week of first exposure and age of the participant accounted for these regional differences. It was found that offspring with VEC were usually exposed in utero to larger total doses of DES administered over longer durations and beginning at an earlier date in gestation than were offspring without these changes. The occurance of VEC is primarily associated with exposure initiated during the first 18 weeks of gestation. The frequency of VEC appears to decrease among older woman. Reference [4] found that, after adjusting for the pattern of DES exposure, the frequency of VEC appears to diminish with age, occurring less frequently among women over 26 years of age. This seems to indicate that VEC remisses with age*. No evidence has yet been produced to show that VEC progresses to adenocarcinoma.

Another DESAD study [7] analyzed the fertility and pregnancy outcome in DES exposed daughters. This study showed no statistical difference in fertility rates between DES daughters and a control group, but did find a statistical difference in adverse pregnancy outcomes (miscarriages, still births and

* Telephone conversation with Dr. Kenneth Noller of the Mayo Clinic

premature births). DES daughters seem to have a higher risk of an adverse pregnancy outcome.

3.2.2 Mothers

Because reproductive factors and ovarian hormones have been implicated in the origin of breast cancer, concern was shown for the development of breast cancer in DES mothers. Laboratory animal experimentation has also revealed the carcinogenic potential of estrogens, including DES. Therefore, a study was performed to determine the risk of cancer in DES mothers [8]. This study found a greater number of breast cancer cases in DES mothers compared to a control group, but no statistical significance could be placed in the result, i.e., the difference can be attributed to chance alone. Another study [9] performed at the Mayo Clinic found no difference in the number of breast or gynecologic cancer cases among mothers treated with DES during pregnancy when compared to the expected incidence. The HHS Task Force therefore concluded that the association between DES and cancer of the reporductive organs in mothers is not established, though it is still suspect.

3.2.3 Sons

Reports have been made that DES sons exposed in utero have a larger number of abnormalities in the genital and possibly lower urinary tract than non-exposed sons, a higher incidence of testicular cancer, and possibly a greater risk of sterility. The HHS Task Force could not find any evidence of an association between in utero exposure to DES and an increased risk of testicular cancer. The Task Force found unresolved questions in studies dealing with the abnormality and sterility questions and requested further research be performed.

3.3 Age-Incidence of Adenocarcinoma and Estrogen Use

After the first medical reports confirmed the association of DES to adenocarcinoma, studies were performed at several institutions to estimate the population at risk and the risk of developing adenocarcinoma. These studies, though rough and incomplete, in addition to the evaluation of the Registry of Clear Cell Adenocarcinoma of the Genital Tract in Young Females provide a view of the age incidence of adenocarcinoma in DES daughters and the extent of use of DES, and related estrogens. The Registry was initiated after the association between DES and adenocarcinoma was established in order to develop information pertaining to the disease. An analysis of the Registry was made in 1977 [10] to determine the incidence rate of adenocarcinoma in DES exposed daughters.

The analysis, which was based on cases reported to the Registry as of February, 1976, shows that the disease usually strikes women between the ages of 14 and 24. The peak age of diagnosis of the disease was 19. Only one case was reported below the range, 7 years of age, and one case above the range, 27 years of age. Total dosage of DES and related estrogens varied widely, ranging from 135 to 18,200 mg. The maximum daily dose also varied widely, from 1.5 to 150 mg in the first three months of pregnancy. Significantly, in all cases in which the time of initiation of treatment was known accurately, treatment began before the eighteenth week of pregnancy. Further analysis revealed that women born in the 1951 to 1953 period have higher incidence rates than those women born in any other previous or subsequent 3 year period, though it should be noted that women born in the 1960's have not all passed the age at risk range. But the data that is available suggests that the prevalence of pregnancy related use of DES was at a peak in the early 1950's. Other studies seem to confirm this conclusion. Based on the rate of DES usage of from 1 to 10 percent in all pregnancies, the incidence rate of

adenocarcinoma in DES exposed females through age 24 was determined to be between .14 and 1.4 per thousand. The authors note that this estimate may be low because the Registry is incomplete and because 10 percent of Registry cases had a maternal history of unidentified medication during pregnancy and were excluded from the calculations, even though it was likely that many were exposed to DES.

The Mayo Clinic has examined records of all children delivered at the hospital between the years 1943 and 1959 to determine incidence rates [11]. They determine the incidence rate to be no greater than 4 per 1000. Over the 17 year period estrogens (93 percent of which was DES) were administered in 7 percent of all deliveries, but usage varied in individual years. From a low of administering estrogens to 2 percent of all deliveries in 1943, usage increased to a high of 19 percent in 1947, decreased substantially to 7 percent in 1951, and then gradually decreased to 2 percent in 1959. Table 3.1 shows this pattern of use.

Of the 1719 children 126 were known to have died. Thirty-eight were stillborn, and another 61 died within the first year, or 5.7 percent of all deliveries. No children were reported to have died from adenocarcinoma. Based on an average 7 percent estrogen use over this 17 year period, the Mayo Clinic estimated that between 1 and 2 million women nationwide were exposed in utero to DES and related estrogens. This figure is not definitive because use varied regionally, and some areas did not use estrogens as much as did the Mayo Clinic.* Neverthless, the figure is the only available estimate for the period.

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* Telephone conversation with Dr. Kenneth Noller of the Mayo Clinic.

TABLE 3.1

TOTAL NUMBER OF CHILDREN DELIVERED
AND PROPORTION EXPOSED TO
ESTROGEN IN UTERO

Year	Total Delivered	Exposed to Estrogen In Utero	
		No.	%
1943	801	17	2
1944	915	18	2
1945	871	43	5
1946	1236	120	10
1947	1442	270	19
1948	1455	251	17
1949	1451	224	15
1950	1506	209	14
1951	1555	102	7
1952	1648	96	6
1953	1593	89	6
1954	1702	60	4
1955	1527	52	3
1956	1393	61	4
1957	1574	47	3
1958	1544	33	2
1959	1759	27	2
Total	23,482	1,719	7

Source: Reference 10

The Boston Collaborative Drug Surveillance Program of the Boston University Medical Center reported on DES exposure between the years 1959 to 1965 in 12 hospitals located across the country [12]. Of the 51,071 pregnancies 217 women received DES (.42%). The pattern of use varied across the country as can been seen in Table 3.2. In all hospitals in the study the frequency of exposure to DES remained reasonably stable between the years 1959 and 1965.

Data was also gathered from 2 market survey organizations, Lea, Inc. and R.A. Gosselin and Co. Lea, Inc. obtains information from 1500 private physicians who report on all pharmaceutical products they prescribe. Gosselin and Co. survey new and refill prescription data collected on a 2 week basis from 400 randomly selected pharmacies throughout the United States. Both of these sources showed about 2.5 million DES prescriptions written per year in 1964 to 1970. Lea, Inc. also obtains the reason for the use of the drug. Between 1960 and 1970 and average of 100,000 DES prescriptions per year were written for pregnant women. There was regionally variation. The drug was more commonly prescribed for prenatal care in the East and Midwest than in the South and West. The maximum number of exposed women in utero, then could be no greater than 50,000 per year. But since from 3 to 5 prescriptions would be prescribed per pregnancy, the number of liveborn female offspring exposed to DES in utero is estimated to be between 10,000 and 16,000 per year in the United States during the period 1959 to 1965.

Therefore between the years 1943 and 1970 a rough estimate of the total number of females exposed in utero to DES and related drugs is between 1.1 and 2.2 million. It has also been established that there was regionally variation in DES use as well as variation in dosage administered. Drug usage probably reached a peak in the early 50's and declined thereafter, prescribed in not

TABLE 3.2

DES RECIPIENTS BY HOSPITAL

1959-1965

HOSPITAL	No. of DES Recipients	No. of Pregnancies	% of DES Recipients
Boston Lying-In	174	11,455	1.5%
Children's-Buffalo	19	2,335	.8
Charity Hospital of Louisiana-New Orleans	2	2,526	.08
Columbia-Presbyterian- New York	0	2,071	0
John Hopkins-Baltimore	0	3,460	0
Medical College of Virginia-Richmond	4	3,131	.1
University of Minnesota - Minneapolis	3	2,949	.1
New York Medical College Metropolitan - New York	2	4,376	.05
University of Oregon-Portland	3	3,014	.1
Pennsylvania, Philadelphia	7	9,571	.07
Prodivence-Lying In	1	2,724	.04
University of Tennessee - Memphis	2	3,459	.06
	217	51,071	.42%

Reference 12

more than 1 percent of pregnancies in the 1960's. This indicates that reported cases of clear-cell adenocarcinoma of the vagina and cervix should have peaked in the early to mid 1970's.

3.4 Estimated Number of Adenocarcinoma Cases

It should be stated at the outset that any estimate of the potential number of adenocarcinoma cases must by nature of the available statistics be rough. No nationwide systematic sample has been taken of any of the necessary variables. Statistics that are available have been developed on the basis of a relatively few hospitals. Any national extrapolation of these statistics must therefore be considered as rough approximations.

Table 3.3 is an attempt to use the available facts and statistics to predict the number of women exposed in utero to estrogens by year of birth, and to predict the number of potential adenocarcinoma cases by year of diagnosis, assuming that diagnosis is always made at the peak incidence age of 19. The table uses the Mayo Clinic estimate of 1 to 2 million exposed daughters born between 1943 and 1959, and the frequency of estrogen use at the hospital for those years to predict the number of exposed daughters by year of birth. An adenocarcinoma rate of 1 per 1000 exposed women was applied to estimate the number of cases expected per year. From 1960 to 1970 the number of exposed daughters was taken directly from study [12]. Column (1) was computed from the Mayo Clinic study [11] and shows the percentage of women exposed by year of birth. Column (3) shows the absolute number of exposed women by year of birth. Column (5) shows the estimated number of adenocarcinoma cases diagnosed in the year indicated in Column (4).

Since we know from the analysis of the Registry cases that adenocarcinoma primaril occurs between the ages of 14 and 24, we can introduce this fact into Table 3.3 to make it more realistic. The frequency distribution developed from the

TABLE 3.3
ESTIMATED ADENOCARCINOMA CASES BY
YEAR OF BIRTH AND YEAR OF DIAGNOSIS

BIRTH YEAR	(1) % DISTRIBUTION USING DES	(2) TOTAL EXPOSED	(3) NUMBER EXPOSED IN (000)	(4) YEAR DIAGNOSED[1]	(5) ADENOCARCINOMA CASES[2]
1943	1.0%		10-20	1962	10-20
1944	1.0		10-20	1963	10-20
1945	2.5		25-50	1964	25-50
1946	7.0		70-140	1965	70-140
1947	15.7	1-2 [redacted]	157-314	1966	157-314
1948	14.6		146-292	1967	146-292
1949	13.0		130-260	1968	130-260
1950	12.2		122-244	1969	122-244
1951	5.9		59-118	1970	59-118
1952	5.6		56-112	1971	56-112
1953	5.2		52-104	1972	52-104
1954	3.5		35-70	1973	35-70
1955	3.0		30-60	1974	30-60
1956	3.5		35-70	1975	35-70
1957	2.7		54-108	1976	54-108
1958	1.9		19-38	1977	19-38
1959	1.6		16-32	1978	16-32
1960			10-16	1979	10-16
1961			10-16	1980	10-16
1962			10-16	1981	10-16
1963			10-16	1982	10-16
1964			10-16	1983	10-16
1965			10-16	1984	10-16
1966			10-16	1985	10-16
1967			10-16	1986	10-16
1968			10-16	1987	10-16
1969			10-16	1988	10-16
1970			10-16	1989	10-16

1. Assuming all cases diagnosed at age 19.
2. At incidence rate of 1 per 1000.

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Registry cases showed the incidence rate of 19 year old exposed women to be approximately 8 times that of exposed women who were 14 or 24 years old. An approximate percentage frequency distribution by age of diagnosis can be computed and is shown in Table 3.4.

Table 3.4

APPROXIMATE PERCENTAGE DISTRIBUTION OF ADENOCARCINOMA CASES BY AGE

AGE	PERCENTAGE OF CASES
14	2.3%
15	4.6
16	8.6
17	10.2
18	12.5
19	18.0
20	13.3
21	12.5
22	10.9
23	4.7
24	2.3

Column (5) of Table 3.3 is multiplied by these percentages to distribute the total estimated cases by year of birth over the 11 year range of diagnosis. For example, the number of daughters born in 1950 estimated to develop adenocarcinoma at age 14 in 1964 is between 3 and 6, and at age 24 in 1974 is between 3 and 6. The total estimated number of cases between 1957 and 1993, developed in this way, is between 1095 and 2226. Table 3.5 indicates that between 1977 and 1993 between 143 and 311 cases can be expected to develop.

Table 3.5
Summary Table
Estimated Adenocarcinoma Cases
By Year of Diagnosis
1977 to 1993

Year of Diagnosis	Number
1977	23-48
1978	21-44
1979	17-36
1980	13-28
1981	10-22
1982	7-17
1983	7-17
1984	7-16
1985	7-16
1986	7-15
1987	7-14
1988	6-12
1989	5-10
1990	3-7
1991	2-5
1992	1-3
1993	0-1
Total	143-311

3.5 Estimated Liability Payments

The ISO Product Liability Closed Claim Survey shows an average total payment of $171,173 for settlements and awards associated with prescription drugs. An inflation adjustment of 35 percent, computed from the Bureau of Labor Statistics Consumer Price Index, was applied to this average payment to bring the dollar figure to May 1980. The inflation adjusted figure is therefore $231,000. The estimated total payments from DES adenocarcinoma cases is shown in Table 3.6.

Table 3.6
Estimated Total Payments
For Potential Adenocarcinoma
Cases

1977 to 1993
(In 1980 dollars)

Cases	Average Total Payment	Total Payments in millions
143-311	$231,000	$33-$71

Adenosis cases have not been included in our estimate because no harmful health effect has been proven, and evidence now points to remission of the effect with age.

A better estimate for potential adenocarcinoma cases would entail adding currently contested court cases to our estimate of future cases. But even if we know the total number of present court cases, we could not use the number. Some present cases are likely to include women who have developed adenosis and these cases would have to be separated from the adenocarcinoma cases. To try to provide a more accurate assessment of potential liability including currently contested cases, we have included all adenocarcinoma cases estimated to have developed from 1970. Table 3.7 provides this estimate. If effect we are assuming that all estimated adenocarcinoma cases developing between 1970 and 1977 are now in the courts.

Table 3.7
Estimated Total Payments
For Adenocarcinoma Cases
Developing From 1970 to
1993
(In 1980 dollars)

Cases	Average Total Payment	Total Payment (In millions)
430-890	$213,000	$91.5-$189.5

Allocated loss adjustment expense average 10.8 percent of payment for payments of $100,000 or more. Loss adjustment expense is therefore likely to be between $9.9 and $20.4 million. Table 3.8 shows the estimated total payments and loss adjustment expenses arising from potential adenocarcinoma cases.

Table 3.8
Estimated Total Payments
and Loss Adjustment Expense
From Adenocarcinoma Cases
Developing From 1970 to 1993
(In 1980 dollars)

	(In millions)
Total Payments	$91.5-$189.5
Loss Adjustment Expense	9.9- 20.4
Total	$100.4-$209.9

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TO THE TASK FORCE ON TORT LIABILITY FOR CUMULATIVE TRAUMA AND LATENT INJURY

Enclosed is the draft preliminary report, "Estimates of Potential Liability from Asbestos and DES Related Injury", which was completed by the Research and Policy Development department for review by the Task Force on September 25.

Sincerely,

E. Palermo
Senior Economist

EP/rs
Enclosure

DEPOSITION EXHIBIT 34 Lewis 5-19-92

34419

ESTIMATES OF POTENTIAL LIABILITY FROM ASBESTOS AND DES RELATED INJURY

Prepared for
Task Force on Tort
Liability for Cumulative
Trauma and Latent Injury

Prepared by
Research and Policy Development
September 17, 1980

E. Palermo

DRAFT

TABLE OF CONTENTS

SUMMARY

This report has attempted to quantify potential product liability payments arising from the deleterious health effects engendered by occupational exposure to asbestos, and the ingestion during pregnancy of diethylstilbestrol (DES).

Asbestos

Asbestos induced cancers and respiratory disease have been confirmed in a number of epidemiologic and animal studies. Lung cancer, mesothelioma, and asbestosis have been the diseases most widely studied and confirmed. Gastrointestinal and laryngeal cancers have also been suggested to have developed from asbestos exposure.

It has also been reported that the magnitude of asbestos exposure increases the degree of risk of developing a disease. But no lower threshold value has been established, i.e., a value below which a disease does not develop in a person. Therefore any person exposed to asbestos for any length of time in a working environment where "asbestos in air" levels are higher than occur in a non-working environment are subject to a higher degree of risk than the general population. Risk levels vary among the many different published studies investigating different occupational groups. In a study conducted jointly by the National Cancer Institute, the National Institute of Environmental Health Sciences, and the National Institute for Occupational Safety and Health, a risk ratio of between 1.5 to 12 times greater than that in the general population was used. Risk ratios determined in various studies lie within this range. Studies have also confirmed a strong synergistic effect of asbestos exposure with smoking.

The latency period within which cancer develops from first exposure is long and the range of years within which it develops is wide. A minimum latency period of 15 years from first exposure for heavily exposed workers has been suggested, and 25 years for less heavily exposed workers. The latency period has been shown to range up to approximately 50 years in some studies.

Most previous studies of the population at risk from asbestos exposure have concentrated on currently exposed workers, and do not separate workers by the duration and magnitude of their exposure. Our analysis has developed a methodology where not only the number of currently exposed workers are estimated, but also former workers who are no longer exposed. In addition the methodology determines the length of their exposure, and an indication of their magnitude of exposure is measured. For former workers the year in which they were removed from exposure is also provided. The 10 industries with the largest number of workers exposed full-time (defined as greater than 4 hours per day) and the 10 industries with the largest number of workers exposed part-time were identified. Because some industries are in both categories 13 manufacturing industries were selected. Contract construction was added to this total because of the large number of exposed workers in the industry, making a total of 14 industries which were finally analyzed. These 14 industries contained 65 percent of all workers exposed to asbestos.

Former employees in these 14 industries were estimated in each year from 1950 to 1979 by number of years of employment. Current employees as of 1979 in these 14 industries were also estimated by number of years of employment. To these two groups the estimated number of WWII shipyard workers were added. For former and current workers the number of lung cancer, mesothelioma cases and asbestosis cases expected to develop between 1977 and 1995 were estimated. To these were added the number of lung cancer and mesothelioma cases expected to develop in surviving WWII shipyard workers between 1977 and 1980 (last year assumed in the latency period). Total payments and product liability payments for settlements and awards were then estimated for these cases.

Table I shows the estimated likely product liability settlements and awards. The table is based on the probability that 50 percent of asbestos induced illnesses will be handled through the product liability system. Average product liability payments were taken from the ISO Product Liability Closed Claim Survey and, after an inflation adjustment, averaged $180,800 for lung cancer and mesothelioma cases, and $190,900 for asbestosis cases.

Table I
ESTIMATED LIKELY PRODUCT LIABILITY PAYMENTS DUE TO ASBESTOS EXPOSURE
1977 to 1995
(In 1980 Dollars)

Illness	Total Cases[1] (In Thousands)	Total Product Liability Payments (In Billions)
Lung cancer & mesothelioma	60.4 - 120.0	$5.5 - $10.8
Asbestosis	30.0 - 41.0	2.9 - 3.9
Total	90.4 - 161.0	$8.4 - $14.7

[1]Based on 14 industries having 65 percent of all exposed members, and WWI shipyard workers. Fifty percent assumed handled by product liability system.

These figures do not include allocated loss adjustment expense which in the Closed Claim Survey averaged 10.8 percent of liability payments. Table II shows likely product liability payments and loss adjustment expenses.

Table II
ESTIMATED LIKELY PRODUCT LIABILITY PAYMENTS AND LOSS ADJUSTMENT EXPENSE DUE TO ASBESTOS EXPOSURE [1]

1977 to 1995
(In 1980 Dollars)

	(In Billions)
Product liability payments	$8.4 - $14.7
Loss adjustment expense	.9 - 1.6
Total	$9.3 - $16.3

[1] Based on 14 industries having 65 percent of all exposed workers and WWII shipyard workers.

The figures presented here on estimated product liability payments are conservative because only 65 percent of all occupationally exposed workers are included. One cannot merely divide the total by .65 to obtain a total estimate because the rate at which workers enter and leave an industry differ, and affects the estimates of former workers in the industry. Individual assessment of the excluded industries would be required to develop an accurate total estimate.

Finally it should be noted that the courts can affect the outcome, if they find a synergistic effect between asbestos exposure and smoking. The establishment of the synergistic relationship between asbestos exposure and smoking may reduce the dollar cost of settlements and awards, if the courts find contributory negligence on the part of smoking asbestos workers.